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HEALTHY MILITARY PERSONNEL

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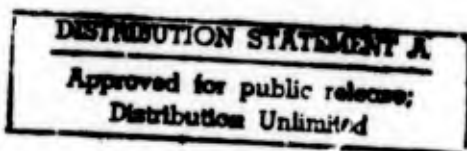
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IDIOPATHIC SPONTANEOUS PNEUMOTHORAX IN APPARENTLY HEALTHY MILITARY PERSONNEL

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IDIOPATHIC SPONTANEOUS PNEUMOTHORAX IN APPARENTLY HEALTHY MILITARY PERSONNEL

A pneumothorax is called *spontaneous* when it occurs unexpectedly, suddenly or slowly. In practice the term covers almost all varieties of pneumothorax other than those resulting from trauma or surgical intervention. The term *idiopathic* or *primary* is applied only to cases occurring in apparently healthy individuals with no evidence of pulmonary disease.

Idiopathic spontaneous pneumothorax (hereinafter referred to as S.P.) is a nosographic category of practical value. In the apparently healthy individual S.P. is a comparatively rare disease, but not quite so rare as was believed in the past.

ETIOLOGY

Earlier, in the evolution of our knowledge, the cause of S.P. was assumed to be tuberculosis unless some other obvious cause could be found. In fact, most reports ascribed S.P. to tuberculosis in 80% to 90% of the cases. Fortunately, with the decline of tuberculosis, S.P. as a complication is now rare.

Kjaergaard (46) probably was the first to emphasize the frequency with which the condition occurs in apparently healthy young persons. He observed only one case of tuberculosis in a followup study of 51 patients with S.P. over a 2-year period. This has been confirmed by an ever-increasing number of cases reported by other authors (27). Perry (78) stated that, out of 250 cases of S.P. mentioned in the literature up to 1939, only 6 patients had tuberculosis.

Undoubtedly it is impossible for a normal pleura to rupture during coughing or from the increased pressure of muscular exertion. During great physical exertion the intrathoracic pressure usually rises about 100 mm. Hg without rupturing or stretching

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lung tissues. Polak and Adams (79) found the rupture pressure of the lung (closed glottis) in mammals (from the mouse to the steer) to be in the order of 60 to 100 mm. Hg. West, cited by Kjaergaard (46), has performed some experiments by inflating lungs *in situ* (postmortem). He found that rupture of the pleura could not be produced until the pressure was above 200 mm. Hg. Such a high intrapulmonary pressure would normally never arise in life. Therefore, one must invariably assume the presence of some other pathologic condition as a prerequisite to the pleural rupture.

Four pathologic entities must be considered in the etiology of S.P.:

1. Pleural adhesions causing the formation of subpleural blebs.

2. Congenital pulmonary cysts.

3. Scar tissue vesicles in the apices of the lungs presenting a valve-like structure.

4. Emphysematous valve vesicles.

Spontaneous rupture of subpleural blebs or bullae and of the visceral pleura is now accepted as the most common cause of S.P. In this so-called "benign" variety of S.P., the subpleural blebs or bullae may be either large or small, single or multiple. More commonly, the pathologic picture is that of one or more small subpleural blebs (localized emphysema) situated in the apex of the upper lobe. Sometimes, a thickening of the visceral pleura in this region may be present. As a rule, however, patients with S.P. do not suffer from generalized emphysema (27). Congenital pulmonary cysts may occasionally cause S.P. Although most cases of benign S.P. actually are likely to be due to ruptured blebs (bullae), some may be caused by the tearing of pleural adhesions; the term *benign S.P.*, however, covers both possibilities.

Hayashi (36) and Fischer (30), as well as Kjaergaard (46) have affirmed that scar formation around a bronchiole of a peripheral alveolus acts as a check-valve, producing inflation of a subpleural bleb but preventing the egress of air.

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Macklin (65) was able to demonstrate in animals that an artificially induced increase of the intrabronchial pressure produces small but widespread ruptures in the walls of the alveoli overlying the finer ramifications of the pulmonary vessels. These ruptures result in the escape of air into the surrounding connective tissue and allow air to move along the fascial planes toward the mediastinum or toward the visceral pleura. If the air crowds to the pleura, it may produce a subpleural bleb or rupture of the pleura, and result in an S.P. But air can also reach the hilar structures via the vascular sheaths of the lung, spreading out into the mediastinum and causing a pneumomediastinum. Secondarily, by rupturing or penetrating the mediastinal pleura, it brings about an S.P. Hamman (33) has postulated that in certain instances S.P. may occur as a result of a rupture of the mediastinal pleura when mediastinal emphysema is present.

Other infrequent causes of S.P. are chronic bronchitis, asthma, bronchial carcinoma, neoplastic metastases, and sarcoidosis. Staphylococcal lung abscesses are apt to erode the visceral pleura, particularly in infancy. Granulomata also predispose to pleural bullae which may eventually rupture.

In very rare cases, S.P. may also arise from excessive traction by retractile lung tissue which brings about a gradual, and probably nonproliferative, separation of the visceral pleura from the parietal pleura. This "negative pressure" S.P. may develop without a rupture of a subpleural bleb. Cases induced by air penetrating the permeable pleura have been described by Laennec (55), Sattler (83), and Cardozo (14).

Other gross pathologic findings, frequently associated with blebs and bullae, are loss of lung elasticity, parchment-like texture of lung tissue, and numerous pleural adhesions. All of these changes suggest a fibrotic degeneration of the lung, the result of a pulmonary vascular insufficiency. Moreover, their presence in the apices and not in the lower portions of the lungs suggests a vascular ischemia in the region of the most severe changes. Therefore, it is possible that the blebs and bullae of the lungs are secondary to an underlying peripheral vascular ischemia (89).

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SYMPTOMS, FINDINGS, AND DIAGNOSIS

The onset of S.P. is usually sudden, but may also be undetected and gradual. Complaints are associated with rather pronounced symptoms. Chest pain (pleuritic pain) of varying degrees is the predominant initial symptom. It occurs over the affected side, is aggravated by respiration, may extend to the shoulder, neck, ears (vagus nerve), back, abdomen, or arms, and gradually subsides to a dull ache in a few hours.

Dyspnea and cyanosis may or may not be associated symptoms, depending on the degree of pulmonary collapse. Cough is the other most commonly present symptom. Pallor and evidence of cardiovascular involvement, even to the point of shock, may occur.

In numerous cases the onset of S.P. is gradual. In other cases the initial symptoms are either nonexistent or mild, ranging from a vague discomfort in the chest to absolutely no symptoms.

The difference between patients exhibiting no symptoms or only mild ones (63.5%) and those exhibiting moderate to severe symptoms (36.5%) is statistically significant (57).

On the average, a patient with S.P. normally has a moderate amount of chest discomfort, some dyspnea on effort, and perhaps cough. Therefore, many cases have been overlooked and misinterpreted as myalgia, pleurisy, or angina pectoris because of complaints similar to those related to a small S.P. (63). Generally, there is no close connection between the severity of the symptoms at the onset of the attacks and the degree of pulmonary collapse (i.e., the size of S.P.). There seems to be a greater frequency of occurrence in the right side over the left one.

Physical examination reveals an absence of breath sounds or a diminution in their intensity over the affected side, and percussion may reveal hyperresonance or tympany when more than 50% collapse has occurred. In these cases the respiration causes less chest wall excursion; the interspaces are not as prominent and the supraclavicular space may also be obliterated by the increased pleural pressure.

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Chest x-rays will establish the diagnosis in most instances. The chest film (posterior-anterior, lateral, and lordotic) ordinarily shows the pneumothorax and a shift of the mediastinal structures, but usually does not reveal any lung disease or pleural effusion. Full expiration will aid in demonstrating a small pneumothorax because the less aerated lung will be denser, and even a small pneumothorax will be more easily differentiated.

The important point in the diagnosis is the correct differentiation of a small S.P. from such conditions as (1) large, thin-walled, air-containing cysts of the lung, (2) pulmonary cavities, (3) diaphragmatic hernia, (4) gas in a distended stomach, and (5) a subphrenic abscess containing gas.

In cases of S.P. there are some diagnostic measures that should be considered: (1) a tuberculin test should be made; (2) after the lung has partially or completely re-expanded special x-ray studies and laminograms are of value in finding cystic or bullous areas in the apex of the lung; (3) bronchoscopy may be useful for diagnosing an endobronchial lesion, especially in cases of recurrent S.P. and when re-expansion of the lung fails to occur following therapy.

COURSE

S.P. occurs chiefly in young individuals—more often in men than in women, and more frequently on the right side than on the left.

The onset of symptoms sometimes coincides with physical exertion, but it is not usually related to physical activities. If this were the case one would expect the disease to occur more frequently in those performing strenuous manual labor, among athletes, or in conjunction with paroxysms of coughing; however, this assumption was invalidated by several studies. In most instances S.P. occurs at rest.

In general, S.P. is relatively harmless and the course of the disease is benign. The collapsed lung usually re-expands satisfactorily and does not collapse again. Full recovery is the rule, unless complications occur.

COMPLICATIONS

In a significant number of instances, complications such as effusion, tension pneumothorax, simultaneous bilateral pneumothorax, hemopneumothorax with severe bleeding, and recurrent and chronic pneumothorax (unexpandable lung) do occur.

Tension pneumothorax

This disease is characterized by its progressive increase in severity up to extreme dyspnea and cyanosis, a shift of the mediastinum to the opposite side, and in some cases, downward displacement of the diaphragm, causing respiratory and circulatory distress. According to DuBose et al. (25), Niehaus (72), and Rottenberg and Golden (81), tension pneumothorax develops in about 10% to 19% of S.P. cases.

Bilateral simultaneous pneumothorax

Bilateral simultaneous pneumothorax occurs less frequently than tension pneumothorax. Its incidence in clinically encountered cases of S.P. varies from 1% to 25%, the average being about 5% to 10% (40, 66). The mortality rate has been reported to be about 50% but has dropped considerably in the last few years.

Not so well documented is the incidence of S.P. on the opposite side following an initial attack. Gaensler (31) found this to be as high as 10% to 15% of S.P. cases.

Pleural effusion

This consequence of S.P. was observed in 16% of the cases (64).

Hemopneumothorax

This is another serious complication of S.P. occurring even less frequently than those mentioned earlier (34, 35, 40, 68). Tearing of adhesions is usually considered to be the cause of hemorrhages. The symptoms of S.P. are followed by increasing dyspnea, weakness, and shock. A hemopneumothorax should be suspected whenever the patient presents a hydropneumothorax within a few hours.

Recurrent and chronic pneumothorax

This complication is relatively common. In from 10% to 33% of the reported S.P. cases (16, 18, 57, 72, 81), the disease has been classified as recurrent; the associated disability to the patient is considerable. An S.P. which has persisted for 3 or more months without re-expansion of the lung may be classified as chronic. This complication was observed in about 6% of the cases (10). Four factors are of importance in the failure of the lung to re-expand: (1) pleural adhesions; (2) ruptured cysts (congenital); (3) scar tissue in the wall of a bulla, bleb, or bronchiole; and (4) a constrictive pleural membrane. In Lynn's (64) data on 200 patients the recurrence rate of S.P. was as high as 71% in patients treated conservatively only.

Maxwell (67) believes that, if followed over a lifetime, more than 50% of the untreated patients will show recurrences. Gaensler (31) assembled 1,080 carefully documented cases from the literature as of 1944. He found that 245 patients, or 22.7%, had one or more recurrences. Other studies have shown similar findings.

I have been unable to discover any comprehensive account of large series of recurrent and chronic cases of S.P. Recurrent and chronic cases, however, seem to be more common than hitherto supposed, and they constitute a real and important problem.

INCIDENCE

Accurate statistics as to the true incidence of S.P. are not available, although the not-so-rare incidence of S.P. among otherwise normal, apparently healthy, young persons has stimulated numerous studies of such cases. Thorough investigation of relatively minor complaints has revealed an incidence of S.P. heretofore unrecognized (77). Thus, S.P. probably occurs more frequently than the literature indicates, since not all physicians report their cases and many persons, whose symptoms are mild, do not consult physicians. With relatively few exceptions, most reports have been concerned with small groups of cases. In the past, reports of larger numbers of cases have usually been drawn from specialized clinics such as tuberculosis hospitals. Reports of this type tend to be biased.

Since World War II many surveys have been made within the Armed Forces, especially of admissions to Army, Navy, and Air Force hospitals. In this way, reports of incidence among the apparently healthy male population became more reliable. Since all military personnel have chest x-rays made prior to entering the service and since many have roentgenograms made after entering active duty, the results observed in these several, fairly large groups, are biased in the opposite direction—namely, away from tuberculosis. The true incidence of S.P. in an otherwise healthy population, including females, is still unknown (although S.P. is diagnosed in students and nurses often enough to suggest that many more cases are overlooked in the population at large (11)).

Civilian life

The incidence of civilian hospital admissions for S.P. ranges from 0.027% to 0.32% (71, 47).

Perry (78) reported 85 cases of benign S.P. from the London Hospital during a 14-year period. Perry's (78) survey of the literature included 250 cases of S.P. (6 with pulmonary tuberculosis). Santos and Tanchanco (82) found only 57 cases of S.P. in a review of approximately 50,000 examinations of the chest, an incidence of 0.11%. Ornstein and Lercher (74) reported 58 cases of S.P. (3 with tuberculosis). Babington (3) reported 44 cases, 31 of which were from the literature from 1935 to 1940. Rottenberg and Golden (81) tabulated 570 reported cases of simple S.P. and reported 105 consecutive cases observed at the Columbia Presbyterian Medical Center, New York, from 1930 to 1947. Of these, 97 occurred in apparently healthy individuals. The average age was 30.2 years. The majority were males. Not a single case of S.P. occurred among 14,000 student nurses who were trained at the same center between 1930 and 1947. Myerson (71) presented 100 consecutive unselected cases of S.P. in patients (38 with tuberculosis) admitted to the Boston City Hospital in a 10-year period between 1934 and 1943. During the same period approximately 375,000 patients were admitted to the hospital, giving an incidence of 0.027%, a figure that coincides with that of the series of Cohen and Kinsman (16). Of these, 73.4% were males; the patients ranged in age from 17 to 80 years, an average of

46 years. Lindskog and Halasz (58) summarize in their report a 10-year experience between 1945 and 1955, on 72 patients with S.P. admitted to a hospital. Of these, 62 (86%) were males; the average age was 34 years. The onset of one S.P. occurred while the patient was flying as a passenger in an airplane.

Armed Forces

Several studies were undertaken for the purpose of determining the etiologic and precipitating factors in cases of nontraumatic idiopathic (primary) S.P. among military personnel, as well as among hospitalized and nonhospitalized military personnel. These studies, however, are only of limited statistical value for comparison with the civilian population since servicemen are regularly admitted for conditions that would not necessitate hospitalization in civilian life. S.P. figures for military hospital admissions are therefore considerably higher than those for civilian hospitals (16, 71). It must also be considered that the age and sex incidence of this condition coincides precisely with the former group of patients: healthy young men, between the ages of 20 and 30 years. Since this group of patients is screened for tuberculosis and other pulmonary diseases prior to entering the service, it cannot be considered as a general sample of the whole adult male population. The incidence of S.P. in the military population has been reported to be as high as 1 per 640 hospital admissions in the US Navy (5) and as low as 41 per 185,578 in the US Army (16). Stein et al. (86) reported 5 clinical cases of S.P. occurring in apparently healthy persons while in military services. In 4 cases there was a history of physical effort; in 1 case S.P. occurred at complete rest. No lung disease could be discovered. Pease et al. (77) surveyed the admissions to an Army hospital (Seymour Johnson Field) and found only 7 cases during twelve months (2 with exposure to tuberculosis in their personal histories). The age of the patients ranged from 19 to 23 years. In 1943 there were 873 admissions to all Army hospitals in continental United States for simple benign idiopathic S.P. (77). In 100 selectees for military service, aged 20 to 30 years, Schneider and Reissman (84) studied S.P. which had occurred months to years before induction examination. Recurrence in 21% of the cases happened more often at rest than during physical exertion. Leach (57) gave a report based on a study of 126 patients with 129 episodes of S.P. observed

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TABLE I

Occurrence of spontaneous pneumothorax in civilian life

Year	Author	Years surveyed	Hospital admissions	S.P. cases	S.P. cases (%)	Right side	Left side	Both sides	Cases with recurrence	Male	Female	Age range	Average age	Occurrence on activity	Occurrence at rest	Remarks
1940	Santos	20	50,000	57	.11											X-ray examinations; 90% tuberculosis cases.
1942	Ornstein	12		58		42	16		17	56	2	15-56	28	10	48	No tuberculosis.
1948	Brock	20		71		42	64	9	42	52	19	1-64	26			
	Myers			115		50	49	1	17	98	17	15-64	23			41 with tuberculosis.
1949	Myerson	10	375,000	100	.027					50	50	12-84		22	78	38 with tuberculosis.
	Rottenberg	18	14,000	0		51	40	6	24	87	10	17-71	30	9	88	Student nurses.
1954	Maxwell	2		23					4							Unselected cases.
1957	Lindskog	10		72			3		54			1-78	34	12	60	
1962	Hyde	10	42,000	200		103			30	192	3	18-68	30	6	194	One in flight (passenger).
	Klassen			135	.316				59	98	37	13-60	30			
1965	Lynn	6		106		47	47	12		97	9	1-79	33	13	93	

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among military personnel in the AAF Training Command. Ages ranged from 18 to 41 years, the average being 24 years.

Heath (37), in a survey of 28,000 admissions to an AAF Regional Hospital (Lincoln, Nebr.), found only 10 S.P. cases admitted. Cohen and Kinsman (16) studied 41 cases of S.P. among hospitalized military personnel (aged 20 to 28 years) in a regional hospital (Fort Bragg, N.C.). Records covering a 10-year period (1935-1944) were reviewed. In the 6-year interval, from 1935 to 1940, there were no cases of S.P. In the succeeding 4 years (1941 to 1944), 41 cases were encountered among 185,578 admissions. The incidence of S.P. based on the annual hospital admissions, was practically constant: 0.027% to 0.03%. Active advanced tuberculosis was found in only 2 cases. Moxon (69) in an 18-month period at the US Naval Hospital, Philadelphia, Pa., established the diagnosis of S.P. in 26 patients; their ages ranged from 13 to 41 years. Jones and Lyons (42) evaluated 60 cases of S.P. seen at the US Naval Hospital, Philadelphia, in a 4½-year period (1949 to 1953), among 1,879 patients admitted to the chest service (S.P., 3.72%). The figures were part of a total hospital admissions of 69,415 patients. S.P. presented, therefore, a total incidence of 0.1008% or approximately 1 for every 1,000 patients admitted to a hospital. The age of the patients ranged between 20 and 30 years. At the US Naval Hospital, Portsmouth, Va., Knowles et al. (49) studied 37 cases of S.P. in servicemen, who were treated either by instillation of talc into the pleural space (30 cases) or conservatively (7 cases). Gaensler (31) reported an incidence of 1 per 1,069 admissions (at the Samson AFB Hospital and 5 other hospitals), i.e., 67 cases of S.P. among the first 71,606 admissions. According to the same author, out of 1,080 carefully documented cases collected from the literature since 1944, and not including the cases mentioned above, 245 patients, or 22.7%, have had one or more recurrences.

Baronofsky et al. (5) have found that S.P. was the admitting diagnosis for 1 out of 640 admissions to a naval hospital over a 3-year period. Thirty-nine of the 114 patients (18 to 47 years of age, most being 25 years or younger) were convinced that they had had a similar episode previously (in most cases on the same side; only four on the contralateral side). Kaufman et al. (44) studied readmissions for S.P. at the San Diego US Naval Hospital

among 72,773 admissions over a 3-year period; the average age was 24.4 years.

Thomas (89) made a survey of 9-year admissions (1948 to 1956) to the Tripler Army Hospital, Hawaii. The number of patients admitted each year for treatment of S.P. had sharply risen from 4 to 11 in the years 1948 through 1953, to 47 in 1956. A corresponding increase from 4.4 per 10,000 admissions in 1953 to 17.4 per 10,000 in 1956 was noted. Over a period of 9 years 156 patients were treated for 184 episodes of S.P.

The following comparison may be of particular interest and importance. In 1945, when the US Navy and Marine Corps had a peak average strength of 3,673,855, the diagnosis of S.P. was made only 651 times; 65 (or 10%) of these cases were readmissions, 83 (over 12%) were invalided from the service, and 6 died (69).

Since 1952, exact statistics on the incidence of S.P. within the USAF have been published by the Surgeon General, USAF, in the "Annual Reports of the Medical Service" where S.P. is shown as a special diagnosis category. From 1952 to 1964, the total annual incidence of S.P. within the US Air Force (male average strength: 880,000) ranged between 450 and 655 cases, an incidence of 47 to 78 cases per year per 100,000 or 0.1% to 0.36% of total hospital admissions (42 to 75 admissions per year per 100,000). The average number of days lost ranged between 22.0 and 55.6. Most of the patients (78% to 94%) were 20 to 30 years or younger.

Related to the total strength of the Armed Forces, the comparison between the figures of the US Navy (1945) and those of the US Air Force (1952 to 1964) shows more than a fourfold increase in the incidence of S.P. Therefore, the morbidity associated with this "benign" entity, computed in terms of hospitalization and loss of effective military service, is of major concern. It becomes increasingly important in times of emergency when all available manpower must be utilized to the fullest possible extent.

Air forces

At ground level S.P. is relatively harmless; in aircrews and in flight, however, the problem is far more serious because of

TABLE II

Occurrence of spontaneous pneumothorax in military personnel

Year	Author	Years surveyed	Hospital admissions	S.P. cases	S.P. cases (%)	Right side	Left side	Both sides	Cases with recurrence	Age range	Average age	Occurrence on activity	Occurrence at rest
1943	Stein, USA	6		5		5				18-30		4	1
1944	Cohen, USA	4	185,578	41	.03	21	20		4	20-30	24	18	23
1945	Pease, USA	1		7		2	5		19	19-23	21	3	4
	Schneider, USA			100		55	44	1		18-38		30	70
	Leach, USAAF			127		76	50	1	3	18-47	24.8	53	72
1946	Heath, USAAF		28,000	10	.034				6				
1950	Moxon, USN	1.5		26					30	18-41	26	3	23
1951	Paul, USA	8		30					1				
1952	Kreutzer, USVA			10					13				
1954	Jones, USN	4.5	69,415	70	.1	35	35		14	17-71	26	8	62
1956	Gaensler, USAF		71,606	67	.09								
1957	Knowles, USN			37									
1958	Kaufman/Baronofsky, USN	3	72,773	114	.15	61	53		43	1-73	24		
	Joynt, RCAF*	10		26									
1959	Thomas, USA	9	235,000	184	.124				28		22		

*S.P. in RCAF = 0.24 per 1,000 (in RAF = 0.22 per 1,000).

the additional hazards and risks of flying. If S.P. occurs at altitude, the already existing problem of hypoxia is greatly magnified. It may result in the abortion of the mission, in a serious aircraft accident, or in a major disaster. Because of its importance in aviation, numerous efforts have been made to clarify the occurrence and frequency of S.P. among flying personnel and, so far as possible, to detect those individuals likely to develop it.

There is insufficient evidence in the literature to suggest that the occurrence of S.P.—or its recurrence—is increased by aerial ascent, rapid decompression, high G-loads, anti-G-suit action, or positive pressure breathing.

In Leach's series (57) of 126 cases of S.P. occurring in young soldiers, 41 were flying personnel, but no episode of S.P. occurred during aerial flight. Of 129 S.P.'s occurring in these 126 individuals, it was suspected that only 3 episodes might have been related to an altitude chamber flight. Holter and Horwitz (39) reported one case of suddenly developed S.P. in a 21-year-old soldier in which the precipitating factor seemed to have been the change of atmospheric pressure produced by ascent to 8,000 feet in an airplane. Heath (37) surveyed the records of 86,916 man-flights in the altitude chamber. The subjects were healthy young crewmen of combat bombers. S.P. occurred in only one instance. In another subject, symptoms due to a pre-existing partial pneumothorax necessitated descent from 17,000 feet. A further survey of 771 individuals undergoing a rapid decompression flight showed no instance of S.P. These findings were in accordance with most data published on rapid decompression up to 1946. Baudot (6) reported 2 other cases of in-flight S.P., neither occurring in solo type of high-performance aircraft, and 1 case that occurred in an altitude chamber. Kircher and Swartzel (45) reported 35 cases of S.P. seen and treated at the Lowry AFB Hospital over a $2\frac{1}{2}$-year period (1951-1953). There was no consistent history of stress or strain at the time of onset. No patient was engaged in flying; only one patient suffered his first episode of S.P. in an altitude chamber (18,000 feet).

Amdur (1) presented a case of a 34-year-old B-47 aircraft observer with recurrent episodes of S.P. due to decreased barometric pressure as a result of actual or simulated flights at

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altitudes above 12,000 to 15,000 feet. With the aid of chest x-rays made before, during, and after altitude chamber flights, a pneumothorax was demonstrated which absorbed quickly at ground level. Markovits and Phillips (66) reported a case of S.P. experienced by a 29-year-old pilot while performing a power dive in a high-performance jet aircraft. Zarriello and Acker (97) reported a case of S.P. occurring in-flight to a 27-year-old Navy pilot. These authors reveal the incidence of S.P. as a fairly common occurrence, but they consider it uncommon in individuals exposed to reduced atmospheric pressures as found in altitude chambers or in flight. For three years (1955-1957) 17,182 young men at the US Naval School of Aviation Medicine have been decompressed in altitude chambers up to a pressure equivalent to 30,000 to 35,000 feet with no known occurrence of S.P. Within the Royal Canadian Air Force (RCAF), Joynt and Laird (43) have observed an incidence of S.P. of 0.24 per 1,000 over a 10-year period. These authors reported 26 cases of S.P. in flyers, with only 1 case occurring during flight. Rumball, cited by Joynt and Laird (43), reported 142 cases of S.P. within the Royal Air Force (RAF)—an incidence of 0.22 per 1,000 over a 6-year period. The recurrence rate was 24.5% over a 5-year followup. Three percent had had bilateral S.P.; 5 cases of S.P. occurred during flight, 28 during severe exertion, 80 at rest. At the USAF School of Aerospace Medicine, Dermksian and Lamb (19) studied 25 flying personnel who had experienced 38 episodes of S.P. with no earlier disorder. Only 3 (12%) of these individuals had had in-flight episodes of S.P. They suffered 11 occurrences of S.P. (29%), all at altitude, either in an aircraft (7) or in an altitude chamber (4); in one of these individuals, the first 2 episodes occurred in flight, and the subsequent ones occurred in the altitude chamber during diagnostic studies at simulated altitudes. At the School of Aerospace Medicine there have been approximately 10,000 man-flights in the altitude chamber over a 5-year period from 1954 to 1958. Of these, 4,000 man-flights included rapid decompression. In these series, there were only 2 suspected cases of S.P.; however, neither was confirmed by diagnostic studies.

Berry and King (9) reported one case of a 34-year-old pilot with 1,780 hours of military flying time, who was referred for evaluation of a suspected S.P. X-rays taken in the altitude chamber demonstrated blebs in the right upper lobe which expanded between

ground level and 30,000 feet. The patient was operated on for an apical wedge resection. He later underwent ascent and rapid decompression in an altitude chamber without any difficulty and returned to flying. Onesti et al. (73) reported a case of a 26-year-old pilot with five years of flying experience who developed idiopathic S.P. during flight. The first sign of the disorder appeared during a rapid descent from 30,000 to 18,000 feet. It is postulated that the positive acceleration to which the pilot was subjected during descent could have caused S.P. Cox and Keshishian (17) reported 77 admissions at the Malcolm Grow USAF Clinical Center at Andrews AFB coded as S.P. over a 4-year period. Most of them were not associated with any strenuous physical activity, and none occurred during aerial flight or chamber flight. Dille (20) recently presented a review on pulmonary disease in general aviation. Among 228,000 active pilots with third class certificates he found that 143 had a history of S.P. Few, if any, had been rejected. This author knows of no case of S.P. that has occurred in flight in civil aviation.

German Air Force

Data obtained over a 10-year period (1957 to 1966) on 15,222 man-flights in altitude chambers were surveyed for the occurrence of S.P. These data concerned healthy pilots, aircrew members (officers and NCO's) as well as Air Force Academy cadets and NCO's undergoing flying training. The flights consisted of 90-minute runs from ground level (atmospheric pressure, about 720 mm. Hg) to a simulated altitude of 42,000 feet (pressure, 127.9 mm. Hg). The rate of ascent was approximately 2,000 to 3,000 feet per minute, and peak altitude was maintained for 15 minutes. The number of individuals exposed to rapid decompression immediately after regular descent from altitude was 13,446. This involved a change in altitude from 8,000 feet (564.4 mm. Hg) to 23,000 feet (307.4 mm. Hg) in less than 1 second, simulating the effect of decompression of a pressurized aircraft flying at 20,000 feet. In these 15,222 man-flights and in the 13,446 rapid decompression runs there were no instances of S.P. except for one person who presented chokes and a circulatory collapse. In this case x-rays evidenced an interlobal pleural effusion; a small, limited S.P. had possibly occurred.

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In the same 10-year period among 24,500 individuals undergoing x-ray examinations of the chest at the German Air Force Institute of Aviation Medicine, no S.P. was found. This review covers more than 64,000 roentgenographic examinations (chest films, laminograms, special examinations).

Three cases of S.P. among GAF aircrew members occurred during the same period—all during rest, none due to strenuous activity, none during flight. The roentgenographic examination (laminograms) did not reveal, in any one case, the presence of emphysematous bullae or cystic degeneration of the lung. After conservative treatment and aeromedical examination, all pilots returned to flying status, with waiver.

AIR TRANSPORT OF PATIENTS WITH PNEUMOTHORAX

A pre-existing pneumothorax will be aggravated upon aerial ascent. The approximate extent to which the trapped air could expand can be determined by applying Boyle's law. The volume occupied by a given quantity of gas is inversely proportional to the absolute pressure exerted on it. The changes in volume occur at comparatively low altitudes: for example, air saturated with water vapor at body temperature (37° C.) at sea level would expand 1.5 times at an altitude of 10,000 feet. The trapped air is free to expand until the pressures are equalized on either side of the tissues. Consequently, the air volume within the pleural space varies inversely to the absolute pressure of the atmosphere. Therefore, a given pneumothorax at ground level will produce increasing degrees of lung collapse during ascent.

A patient with a small pneumothorax may be transported by air on a flight which does not exceed an altitude of 6,000 to 10,000 feet. He should not be transported by air in the presence of a large or tension pneumothorax unless the cabin is pressurized to nearly ground level; moreover, the pneumothorax must be reduced by suction before takeoff. The one-way flutter valve has proved its usefulness in these cases, and it should always be available in flight, especially in cases of tension pneumothorax if suction has not been practiced before flight (12, 23, 32, 59-62). Downey and Strickland (24) reviewed 568 flights of patients with nontuberculous pulmonary disorders; of these, 19 experienced S.P., although

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TABLE III

Occurrence of spontaneous pneumothorax in flying personnel

Year	Author	Years surveyed	S. P. cases	Man-flights in altitude chamber	S. P. cases in chamber flights	Rapid decompression man-flight	S. P. cases in aerial flights	Cases with recurrence	Permanently grounded	Returned to flying duty	Treatment	Remarks
1945	Holter, USN Leach, USAAF		41		3		1					Alt. 8,000 ft.; aircrewman 21 yr.
1946	Heath, USAAF			86,916	2	771	1		29	12		Alt. 30,000 and 15,000 ft.; 2 aircrewmen 19 yr.
1949	Baudot, FAF	8	2		1							Several pilots included.
1951	Paul, USA		30									Pilot 29 yr.
1954	Kircher, USAF	2.5	35		1			5				
1956	Amdur, USAF		1		1			1				Recurrence in chamber flights 13,000 to 17,000 ft.; pilot 34 yr.

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Year	Author									Notes
1957	Markovits, USN		‡			1	1			Pilot 29 yr.
1958	Joynt, RCAF	10	26			1	1			
	Rumball, RAF	6	142			5	24.5%		26§	26 jet pilots. S.P. = 0.24/1,000. S.P. = 0.22/1,000.
1959	Berry, USAF			1‖	10,000				1	Apical wedge resection.
	Dermksian, USAF	10	25	2**	4,000	3††	8		5	
	Stonehill, USAF		1	0	17,182	1	1			Pilot 34 yr.
	Zariello, USN	3	1			1				Pilot 27 yr.
1961	Burgess, RCAF	6	11			1			11	Kaolin pleurodesis.
	Onesti		1			1				Pilot 29 yr.
	Tillisch					1				Dubious; pilot 31 yr.
1963	Dominy, USAF		91				91	‡‡	91	Surgical treatment.
1964	Cox, USAF	4	77						77	26 bed rest; 8 aspiration; 36 tube thoracotomy; 12 definitive surgery.
1966	Dille, FAA*		143		15,222			44		Active pilots, none grounded.
1967	Fuchs, GAF†	10	3	1**	13,446			38§§		

*288,000 pilots.
†24,500 aircrewmen.
‡No S.P. cases.
§Kaolin pleurodesis.
‖Doubtful S.P. Apical blebs enlarged. Pilot 34 years.
**Dubious.
††Aged 27, 30, 34 years.
‡‡Not noted.
§§By waiver, after conservative treatment.

none noted symptoms. These authors recommend a maximum altitude of 10,000 feet for the air transportation of patients with pneumothorax.

TREATMENT

In general the course of S.P. is benign, and complete recovery is the rule. Nevertheless, long periods of disability may occur, the patients may require hospitalization for a long time, and serious complications may develop. It is important, therefore, to institute proper treatment so that the patient may return to work as soon as possible with a reasonable chance of a permanent cure. The treatment should have a very low risk (45).

The purpose of the treatment is to attain re-expansion of the lung by closure of the ruptured subpleural bleb and visceral pleura. The majority of the patients will satisfactorily seal the rupture and re-expand the lung within a few days to a few weeks by conservative management. This care should consist of bed rest, lying on the affected side or prone with head and chest lower than pelvis and feet to avoid any tension of apical pleural adhesions, and needle aspiration as necessary. If dyspnea is uncomfortable, oxygen by nasal catheter or mask may be employed; if pain and cough are distressing, sedatives and analgesics may be used. No respiratory depressors should be used. Usually, with this management the patient exhibits progressive re-expansion of his collapsed lung.

If the amount of the patient's lung collapse does not change within one week, then water-sealed negative drainage (negative pressure of not more than 10 to 15 cc. water, depending on the re-expansion rate) should be instituted. Experience has shown that if expansion has not occurred by this time, the chances of complications (pleural effusion, empyema, fibrosis of pleura, and nonexpandable lung) are greatly increased. Antibiotics should be administered during the suction period. Suction drainage, however, is dictated from the onset of S.P. in cases of (1) tension pneumothorax; (2) a physiologically impaired contralateral lung; and (3) associated cardiac disease with marked dyspnea.

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If the lung does not re-expand by these means within one week, thoracotomy, surgical removal of blebs or partial (wedge) resection, segmental resection and technics to induce symphysis of the pleura over the blebs (rubbing, touching with silver nitrate, etc.) or partial parietal pleurectomy should be done without further delay. Experiments to produce a pleurodesis by artificial pleuritis have not always been successful; in some cases, the loss of the mobility of the chest wall and the diaphragm has had harmful effects upon lung function. Such experiments include pleurisies induced chemically by gomenol oil, or caustic agents such as dextrose, silver nitrate, iodized talcum, guaiacol, iodoform, and poudrage with kaolin, as well as irritation of the pleura by mechanical means such as rubbing the parietal pleura with a gauze sponge. Pleurodesis by surgery of the blebs and by pleurectomy gives the best results—preventing further recurrences, functional damage, and loss of time.

CURRENT POLICY

The problem of S.P. is serious enough in normal occupations; in aviation it becomes more critical because of the victim's possible incapacitation and subsequent inability to control the aircraft (66). The consequences may be loss of life and materiel and abortion of important missions.

As of August 1957, the US Air Force and the US Navy withhold aviators from further flying because of a history of S.P. unless the flyer has had only a single, isolated incident.

Individuals with a single episode of S.P. may be considered for a waiver if evaluation shows complete recovery with full re-expansion of the lung, and there is no demonstrated pathology which would predispose to recurrence. If these conditions are not met, return to flying duties may be considered only after a successful pleurodesis and demonstration of normal pulmonary function after an observation period of six months. All such cases must be given an evaluation in a low pressure chamber, with a medical officer, aerospace medicine, in attendance. Otherwise the flyer will be permanently grounded (AFM 160-1, ch. 5, pars. 76d(3) and e(2)).

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S. P., history of S.P. within the last three years, or history of recurrent S.P. authenticated by hospital report or statement of attending physician, are causes for rejection for enlistment, commission, and entrance to the Air Force Academy, unless there is proof of successful pleurodesis (artificial obliteration of the involved pleural space) with no subsequent episode for two years (AFM 160-1, ch. 5, pars. 76a(4), b, and c).

When an examinee is found to have a disqualifying defect for flying duty such as recurrent S.P., the defect may be waived only by Headquarters US Air Force (AFM 160-1, ch. 3, par. 19e(10)).

Regulations in the Royal Canadian Air Force prohibit high altitude flying by a pilot after a single episode of S.P. and require that a co-pilot be with him when he flies at low altitudes (43). Full flying status has been granted to flyers in the Royal Canadian Air Force following treatment of S.P. if complete pleurodesis has been obtained. Failure to induce an artificial pneumothorax three months after treatment in at least three sites on the affected side has been accepted as proof of obliteration of the pleural space (43).

The British Royal Air Force is advocating pleural symphysis (pleurodesis) or open thoracotomy with pleurectomy before flyers may be returned to flying status. The same policy in cases of S.P. holds within the German Air Force and in all other air forces, so far as I have been able to ascertain.

No dogmatic policy can cover all situations: exceptions concerning individual aspects and service conditions should be possible, but aircrewmen returned to flying status should be restricted to nonsolo flights. This represents a serious reduction of highly trained flying personnel, as well as a considerable economic loss to the Air Force. The cost of training a pilot to full operational activity is $200,000 to $250,000 or more. It would appear important, when possible, to rehabilitate this group to flying status by surgical treatment. The general disposition of persons who experience S.P. while in military service, but not on flying status, is reclassification to limited military duty following one occurrence of S.P. and separation from the service following a simultaneous bilateral or recurrent unilateral episode unless surgical treatment is successful.

DISCUSSION

Recent studies indicate that S.P. is not idiopathic in a strict sense. Radiographic, thoracoscopic, and histologic observations reveal a high incidence of lung surface blebs and bullae in these patients. Therefore, an S.P. should be called "idiopathic" only in those cases in which the etiology cannot be verified.

The incidence of S.P. appears to have noticeably increased. An augmented prevalence of this disease has been noted among the military population during the last decade. This disease predominates among young men of military age.

Although S.P. is neither common nor important statistically as a cause of morbidity and mortality within the Armed Forces in general, in aircrews it is of particular importance.

High-performance aircraft subject the individual, especially his cardiopulmonary system, to great and varied stresses such as high altitudes, rapid decompression, breathing of pure oxygen with forced expiration, increased gravitational forces, and chest constriction due to wearing of anti-G-suit. Any or all of these may conceivably play a role in the pathogenesis of bleb formation or actual rupture with subsequent S.P.

In rapid decompression, the reduction in barometric pressure is transmitted nearly equally and promptly to all body tissues so that the ratio between the intrapulmonary and the intrapleural pressure remains nearly the same. High altitude, likewise, has no effect on the volume or pressure of air- or gas-containing structures so long as they have access to the ambient atmosphere. Changes in pressure and volume will occur only in gas-containing structures that have inadequate or no access to the external atmosphere. So long as there is no air in the pleural space there will be no relative change in intrapleural pressure. With a change in altitude from ground level to 42,000 feet the volume of trapped gas increases 7.6 times (Boyle's law). If the change occurs suddenly as in the event of rapid decompression, the volume of the pressurized compartment, the size of the opening, the pressure differential, and the flight altitude at which decompression takes place must be considered. The rate of decompression is measured in pounds per

square inch per second. Theoretically, the possible amount of expansion of internal body gases at differentials of 1 to 8 p.s.i. per second with change in altitude from 15,000 to 60,000 feet is 29.8 times.

Occurrences of S.P. during flight or simulated flight in an altitude chamber seem to be precipitated by decreased atmospheric pressure: these episodes of S.P. with chest pain did not recur at ground level.

The mechanisms of this phenomenon led to much speculation. It might be understood commonly as (1) rupture of an emphysematous bleb because of relative increase of pressure within the (closed or nearly closed) bleb with rupture of the visceral pleura; (2) rupture of a bleb or an alveolus, thereby causing pulmonary interstitial emphysema with an escape of air to the mediastinum, along the vascular sheaths to the hilus, and then penetration of the frontal mediastinal pleural edge into the pleural space. Roessle, cited by Benzinger (8), found and described the anatomic cause of the severe, rapid decompression sickness as a "pulmonary pneumatosis." Clark (15) reported 2 cases of mediastinal emphysema following rapid decompression of human subjects from a simulated altitude of 8,000 feet to a simulated altitude of 31,000 feet in 0.5 second.

The presence of an abnormal air-containing space in the thorax (cyst, bleb, etc.) in no way handicaps the individual when it has free access to the bronchial tree. When the flyer's condition has been tested in an altitude chamber flight, this kind of lesion is not a contraindication to flying.

The crucial consideration during altitude exposure is the rapidity of the exchange of air or gas between the air space and the tracheobronchial tree. Should this exchange be inadequate, a drop in ambient barometric pressure would cause the gases within the cavity to expand, resulting in either enlargement or rupture. When the air space fails to enlarge, it must be assumed either that flow of air or gas between the space and the tracheobronchial tree is adequate or that the walls of the lesion are rigid enough to withstand the increased pressure.

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If some of the blebs or bullae do not have access to the respiratory tract, it connotes, at altitude, the possibility of expansion to the point of rupture with the inherent dangers to, and incapacitation of, the aircrew member. This possibly explains incidents occurring in flight at less than a ground level ambient pressure.

Air-trapping can also occur when mucous plugs, edema, or inflammation of the bronchioles leading to the air space so alter the ostia that an inadequate transfer exists, with resultant air space rupture at altitude (75). A partial "ball valve mechanism" may also play a role as Amdur (1) suggests: "This assumption is supported by the fact that in one of the chamber flights it was possible, by slow ascent and prolongation of the flight at 19,000 feet, to take the patient to 40,000 feet peak altitude where he experienced no discomfort. Apparently the trapped air may egress into the bronchi provided the rate of ascent and pressure differentials allow equilibrium to take place."

Hayashi (36) and Kjaergaard (46) have suggested that S.P. may also occur at normal atmospheric pressure in this valve-like way—that air entering the bleb (by inspiration) but not escaping by expiration, builds up pressure within the bleb, and rupture occurs. Physical laws, however, should lead us to reject this opinion. Obviously, by opening the "valve," pressure within the bleb would equilibrate with the pressure of the ambient (bronchial) atmosphere. Under the influence of exertion and increased intrathoracic pressure by a closed glottis and a fixed diaphragm (especially under pressure breathing conditions and decreased ambient barometric pressure) any valve mechanism can probably cause the occurrence of S.P.

The fact that the vesicles or blebs observed by Dermksian and Lamb (19) expanded with the progressive decrease in atmospheric pressure, confirms the impression that such vesicles and cysts are air-containing sacs without access to airways. Because it is most difficult to demonstrate abnormalities clinically prior to postmortem or postsurgical examinations, the etiology of S.P. in the apparently healthy young individual is still doubtful.

In spite of Clark's (15) experiences and the one fatal accident observed by Luft (63) within a 10-year period, there have been

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only eight cases of lung damage among thousands of USAF personnel trained in the altitude chambers (AFM 160-5, ch. 6. par. 6-6). It appears unlikely that the usual rapid decompression from 8,000 to 23,000 feet (564.4 mm. Hg = 10.91 p.s.i. to 307.4 mm. Hg = 5.94 p.s.i.) causes significant pulmonary damage unless the glottis is closed or lung pathology exists.

The recent decompression experiments with chimpanzees, performed by Koestler (50), showed no harmful effects of rapid decompression on the lungs. Nineteen chimpanzees, breathing pure oxygen for three hours at an altitude of 600 feet (650 mm. Hg) were first decompressed to an altitude of 35,000 feet (179 mm. Hg) where they remained for 45 minutes. Then they were decompressed unprotected (breathing pure oxygen) to an altitude of 150,000 feet (less than 2 mm. Hg) in 0.8 second and remained at this altitude from 90 to 210 seconds.

Results of these tests, which were of a replicatory nature, have substantiated that denitrogenated chimpanzees can survive sudden exposure to a near vacuum.

Bancroft and Dunn (4) had shown in experimental decompressions of dogs to a near vacuum environment (less than 2 mm. Hg, absolute) that denitrogenation appeared to reduce the incidence of lung damage.

One of Koestler's subjects of questionable fitness expired following 90 seconds' exposure to a near vacuum, but the cause of death in this case was attributed to a failure in the conducting mechanism of the myocardium (50). Because this case represented one of the few opportunities to examine the lungs, immediately after explosive decompression, careful evaluation was made of the smaller bronchioles, alveolar ducts, and alveoli. No indication of disruption or hemorrhage in any of these areas was found.

Of course, the rapid decompression of chimpanzees cannot be extrapolated directly to human beings. The findings, however, imply that rapid decompression, generally, has no harmful effects on lung tissues unless the glottis is closed or pathologic changes exist.

Another potential hazard is associated with the pressure oxygen system required at operational altitudes. In order to prevent hypoxia above 35,000 feet, some method of increasing the tension of the partial pressure of oxygen in the alveoli must be used. One method is to pressurize the lungs by using an oxygen system which delivers oxygen under pressure greater than that of the ambient air. The mask must remain sealed on the face even though positive pressure is applied.

Breathing against pressure entails a reversal of one characteristic of respiration inasmuch as expiration becomes an active (forced) rather than a passive experience. In the instance of apical emphysematous changes, rupture of blebs or bullae followed by S.P. is a potential hazard (22).

Ornstein and Lercher (74) proposed an explanation for the pathogenesis of acquired subpleural blebs and demonstrated a disproportionate overventilation (overdistension) of the lung apices during forced expiration against a closed glottis (Valsalva maneuver) which, if repeated, could rupture the subpleural alveoli resulting in bleb formations. This possibility should be considered in all pilots who have experienced several years of pressure breathing; it also should result in a routine laminogram of the lung apices of these individuals to find out in time the pathologic entity of the apical subpleural bleb formation which causes S.P.

One explanation of the relative rarity of S.P. may also lie in the protection afforded the lung by collateral pathways for the passage of air, as first described by Van Allen et al. (92). This collateral ventilation provides an indirect peripheral pathway for the passage of gases between adjacent lobules and segments when the direct bronchial route of one is occluded. This possibility should be considered in all cases of roentgenologically proved blebs where no evidence of S.P. appears in spite of exposure to extreme conditions.

In recent years, there has accumulated laboratory evidence that the pulmonary effects associated with the gravitational and accelerative forces in flying—such as arterial hypoxia, pulmonary atelectasis, and disruption of pulmonary parenchyma—may be of practical pathophysiologic importance. As flyers undergo high G-loads, the

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intrapleural pressures change as a result of the hydrostatic pressure differences in the pulmonary circulation. With an increase of the hydrostatic pressure in the dependent parts of the lungs, the intrapleural pressure at the dependent surfaces of the lung will be increased. Concomitantly, the intrapleural pressure over the superior surfaces of the lung will become much more negative (up to -32 cm. H_2O). We know that the elastic alveoli are maintained in an expanded position by the negative intrapleural pressure. Because the alveolar pressure remains at ambient pressure, a striking increase in the transalveolar pressure gradient will occur and produce severe overdistension of the alveoli of this region. So, exposure up to 5 to 6+ G levels of forward acceleration occasionally produces structural damage to pulmonary parenchyma (overdistension and rupture of the alveoli), damage to tissues by the development of mediastinal emphysema (experienced by Marshall, undergoing 6+ G), or hemoptysis (95).

Moreover, high G-loads and wearing of anti-G-suits, with the resulting increased intrathoracic pressures, influence the physiology of respiration to some degree. The relationship between S.P. (or pneumomediastinum) and the breathing of 100% oxygen under pressure (increased intrabronchial and intra-alveolar pressure), high G-loads, and anti-G-suit action (increased intrathoracic, vascular, and hydrostatic pressure) requires further study because—

1. Breathing 100% oxygen seems to favor (a) external compression of the lung and (b) changes in the surface-active material (surfactant) lining the alveoli, both causing atelectasis.

2. During prolonged oxygen breathing, nitrogen is eliminated from the body leaving only water vapor, carbon dioxide, and oxygen to exert gas pressure in the lung. It has been shown that the absorption rate of 100% oxygen under 1 atmosphere of pressure is 60 to 80 times greater than the absorption rate of air. Breathing pure oxygen at 258 mm. Hg should speed up this rapid absorption even more. When an airway is obstructed, atelectasis by resorption will occur rapidly.

3. In the normal alveoli pure oxygen is resorbed very fast; in the case of bronchiole obstruction, this fact leads to the so-called

resorptive "aero-atelectasis." In a bleb or bulla—i.e., in a pathologic structure without any resorptive condition—this rapid resorption of oxygen does not occur. Compared to the neighboring alveoli, the pressure in the bleb or bulla, sealed by a temporary "valve," remains higher for a longer period of time. Such a "valve-like mechanism" can also be produced by the hydrostatic (hemodynamic) changes following exposure to increased gravitational forces and chest constriction due to wearing of anti-G-suit, as demonstrated by Wood et al. (94) and by Rahn and Farhi (80).

Suspected cases of S.P. occurring at altitude, diagnosed only by chest pain and not recurring in simulated altitude chamber flight, should be considered as possible cases of "postflight chest pain" (29) or "aero-atelectasis" syndrome (96).

The primary concern in flying personnel who have suffered an S.P. at altitude is the possibility of a complete collapse of the affected lung or the occurrence of a tension pneumothorax. Either complication could severely compromise the ventilatory function of the remaining lung and could embarrass cardiovascular hemodynamics. This would magnify the problems of hypoxia and circulatory stress associated with flying.

In flight, S.P. incapacitates the individual and causes him to relinquish his duties or to abort a mission. Occurring in a single-seated high-performance aircraft, it might result in loss of life and aircraft, and the case may be recorded in the files as an "unexplained" aircraft accident due to human error. Therefore, a conservative attitude prevails in grounding a flyer with pulmonary bullae, blebs, and cysts, and is fully justified unless it can be proved that the air within the lesion is not trapped. One method of investigating the free flow of air into and out of a space is to compare by x-rays the size of this space at ground level and at a simulated altitude.

A young person not required to fly, who collapses from an S.P. for the first time and who responds readily to bed rest, aspiration, or even tube thoracotomy, needs no further treatment. This same patient whose pleura continues to leak after tube thoracotomy and suction needs open definitive surgery. A patient who has had more than two recurrences, although not in a responsible flight

category, should be seriously considered for surgery. The patient's age, past history, type of duty, and degree of responsibility are to be considered. Before an experienced pilot (or aircrew member) is permanently grounded because of an established diagnosis of S.P., he should be given the benefits of modern surgical treatment if a chance for a possible cure exists in his particular case. This decision should be made by the individual pilot, the flight surgeon, and the thoracic surgeon (97).

Conservative therapy is deemed insufficient for aircrews, because of the known prevalence of S.P. recurrence with its attendant dangers in high altitude missions. Before being cleared for unrestricted flying duties, flying personnel must have been cured of their S.P. as shown by subsequent failure to produce a pneumothorax on the affected side artificially (RCAF) or by simulated flight in an altitude chamber (USAF). A pilot who has had one episode, or a patient who has a responsible position in an aircraft (navigator, co-pilot, engineer, paratrooper) must have definitive surgery: corrective surgery removing emphysematous bullae, or a segmental or wedge resection of the diseased area with parietal pleurectomy. Visceral-parietal pleural irritation by the use of silver nitrate or gauze will highly minimize the possibility of a recurrence or the development of other complications. A patient who has not had a pneumothorax but has large cysts visible with x-rays (including laminograms) should also have definitive surgery if he is to stay on flying status. X-ray studies—performed by Dermksian and Lamb (19), Amdur (1), and Dominy and Campbell (22)—in simulated flight in an altitude chamber, showed progressive expansion of air-containing bullae on ascent. This last indication could well be extended to the person who has to fly in the pursuit of his ordinary duties. This program not only eliminates the cause of S.P. attacks, but insures complete symphysis of the pleural surfaces. There is no proof that such symphysis interferes with lung function (64). Modern surgical treatment makes it possible, therefore, to return the majority of S.P. patients to unrestricted flying.

From these reported data, it appears that S.P. is a relatively rare occurrence at altitude. It is logical to conclude that changes of atmospheric pressure alone do not precipitate the incidence of initial attacks of S.P. in individuals with no pre-existing lung disease. The impression that normal aerial flight is likely to produce

S.P. in a healthy individual, as suggested by several authors, must be considered erroneous. The occurrence of S.P. during aerial flight is merely coincidental.

Operational flight, however, may possibly induce occurrence of S.P., both in individuals with pre-existing pulmonary malformations (as blebs, etc.) and in individuals exposed periodically over several years to unusual stresses (high altitude, rapid decompression, pure oxygen pressure breathing with forced expiration, increased gravitational forces, and chest constriction due to the wearing of anti-G-suit)—all of which probably predispose to the development of subpleural blebs.

PNEUMOTHORAX AND AEROEMBOLISM

Pneumothorax, existing before aerial ascent or occurring in flight, leads to severe dangers for the individual. On the other hand, an artificial pneumothorax established before rapid decompression may have a protective effect against aeroembolism as the experiments of Desaga, cited by Benzinger (8), and Kolder (51) have shown. Desaga used a pneumothorax as an air layer around the lungs to protect against blast effects. The test animal (dog) showed subpleural pulmonary changes only in the lower lobes where the air layer was thinnest. The lungs of the control animals, on the contrary, were severely and extensively damaged. Kolder (51) decompressed approximately 700 rats and evaluated the results to clarify the extent to which lethality after rapid decompression is a function of the pressure difference and decompression time. The effects of rapid decompression are directly proportional to the pressure difference and inversely proportional to decompression time. Postmortem findings and postdecompression symptoms are best explained by arterial air embolism. The fact that an artificial pneumothorax offered almost complete protection against rapid decompression led Kolder to suggest that air embolism in rapid decompression is probably not caused by liberation of gases in the blood but only by rupture of alveoli and pulmonary vessels. It could mean that an elastic slackening of lung tissues by a small artificial pneumothorax (or a pneumoperitoneum) may be of protective value in the case of rapid decompression. This hypothesis, although in contrast to the experiences and findings which Koestler (50) substantiated in the rapid decompression

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of chimpanzees, should be considered for further investigation. This is especially the case when a two-gas environment is used in space cabins with its increased danger for the individual undergoing an accidental rapid decompression.

SUMMARY

The medical literature on S.P. is reviewed in regard to etiology, symptomatology, incidence, course and complications, recidivism, and treatment. These reports cover the period from 1940 to 1966, and include, in addition to the civilian population, apparently healthy military personnel (US Army), especially aircrews (USAF, RCAF, RAF).

German Air Force data on 18,225 man-flights in altitude chambers, followed by 13,446 rapid decompression flights over a 10-year period (1957-1966) are surveyed, revealing only 1 case of probable S.P. Three cases of S.P. among GAF aircrew members were recorded in the same period, all during rest, none in flight, or during strenuous activity. The assumption that S.P. is likely to be caused by decreased barometric pressure in aerial ascent, simulated flight, or rapid decompression is an erroneous one, and is not supported by facts. The occurrence of S.P. during normal aerial flight in individuals without definite, pre-existing pulmonary disease is not common enough to be of practical significance. It is apparently no greater than that of idiopathic S.P. occurring during other activities, either in civilian or in military life. But the forces to which a pilot flying high-performance operational aircraft is exposed may produce changes in the lungs favoring the development of S.P. In view of precipitating conditions in flight, such as increase in the intrathoracic pressure by decreased ambient atmospheric pressure, positive pressure breathing, high-G-loads, effects of wearing anti-G-suits, candidates with a previous history of S.P. should be rejected by the aeromedical selection board.

In the case that an active pilot or aircrew member suffers an S.P., the decision to return either to flying status should be based on a thorough aeromedical examination excluding all predisposing conditions for the recurrence of S.P.

If the flyer is returned to flying duty with a waiver, precautions should be taken that he fly neither single-seated high-performance aircraft, nor other aircraft without a co-pilot, and that he avoid maneuvers involving high G-loads unless he has undergone surgical treatment to prevent recurrences of S.P.

The modern approach to S.P. will decrease hospital morbidity and provide a more effective utilization of manpower during periods of total mobilization. In contradistinction to the experience of World War II, patients need not be separated from the service because of recurrent S.P. These individuals may be surgically treated, rehabilitated, and returned to full military service, including flying status in selected instances.

REFERENCES

1. Amdur, R. D. Recurrent spontaneous pneumothorax caused by aerial flight. J. Aviation Med. 27:456 (1956).

2. Artificial pneumothorax and flying. J.A.M.A. 149:1611 (1952).

3. Babington, H. S. Idiopathic spontaneous pneumothorax. Western J. Surg. 52:73 (1944).

4. Bancroft, R. W., and J. E. Dunn, II. Experimental animal decompressions to a near vacuum environment. Aerospace Med. 36:720 (1965).

5. Baronofsky, I. D., H. G. Warden, J. L. Kaufman, J. Whatley, and J. M. Hanner. Bilateral therapy for unilateral spontaneous pneumothorax. J. Thorac. Surg. 34:310 (1957).

6. Baudot, J. Emphyème bulleux et pneumothorax spontané. J. Franc. Med. Chir. Thorac. 3:275 (1949).

7. Benign spontaneous pneumothorax. Brit. Med. J. I:284 (1963).

8. Benzinger, T. Explosive decompression, vol. I, pp. 395-408; and Physiological effects of blast in air and water, vol. II, pp. 1225-1259. German Aviation Medicine in World War II. Washington, D. C.: U. S. Government Printing Office, 1950.

9. Berry, C. A., and A. H. King. Use of altitude chamber in the diagnosis and disposition of problem aeromedical cases. J. Aviation Med. 30:258 (1959).

10. Brewer, L. A. III, Dolley, F. S., and Evans, B. H. The surgical management of chronic "spontaneous" pneumothorax: Report on etiological factors and surgical treatment employed in 15 cases. J. Thorac. Surg. 19:167 (1950).

11. Brock, R. C. Recurrent and chronic spontaneous pneumothorax. Thorax 3:88 (1948).

12. Brown, C. The effect of rapid changes of altitude on patients undergoing pneumotherapy. Dis. Chest 23:175 (1953).

13. Burgess, C. N., and D. G. M. Nelson. Therapy of spontaneous pneumothorax in RCAF flying personnel. Escape and Survival, AGARDOGRAPHY No. 52, New York: Pergamon Press, 1961.

14. Cardozo, E. L. Nonperforative pneumothorax with negative pressure: Traction pneumothorax. Dis. Chest 42:218 (1962).

15. Clark, D. M. Mediastinal emphysema (pneumomediastinum) following explosive decompression of humans. Report of two cases. Memorandum Report TSEAL, AAF Materiel Command (1945).

16. Cohen, S., and J. M. Kinsman. Nontraumatic spontaneous pneumothorax among military personnel. New Eng. J. Med. 235:461 (1946).

17. Cox, P. A., and J. M. Keshishian. Definitive surgical treatment of spontaneous pneumothorax. Aerospace Med. 35:62 (1964).

18. Daughtry, D. C., and J. G. Chesney. Treatment of spontaneous pneumothorax. Nat. Tuberculosis Ass. Tr. 48:66 (1952).

19. Dermksian, G., and L. E. Lamb. Spontaneous pneumothorax in apparently healthy flying personnel. Ann. Intern. Med. 51:39 (1959).

20. Dille, J. R. Pulmonary disease in aviation. Aerospace Med. 37:732 (1966).

21. Dille, J. R., and A. H. Hasbrook. Injuries due to explosion, decompression and impact of a jet transport. Aerospace Med. 37:5 (1966).

22. Dominy, D. E., and D. C. Campbell. Surgically correctable acquired cystic disease of the lung as seen in flying personnel. Dis. Chest 43:240 (1963).

23. Dowd, K. E. Report of death of passenger under treatment by pneumothorax. J. Aviation Med. 16:346 (1945).

24. Downey, V. M., and B. A. Strickland, Jr. Air transportation of cardiac and pulmonary patients. Ann. Intern. Med. 36:525 (1952).

25. DuBose, H. M., H. J. Price, and P. H. Guilfoil. Spontaneous pneumothorax: medical and surgical management. New Eng. J. Med. 248:752 (1953).

26. Dunn, J. E., II, R. W. Bancroft, W. Haymaker, and J. W. Foft. Experimental animal decompressions to less than 2 mm. Hg absolute (pathologic effects). Aerospace Med. 36:725 (1965).

27. Ellis, F. R., Jr., and D. T. Carr. The problem of spontaneous pneumothorax. Med. Clin. N. Amer. 38:1065 (1954).

28. Engel, G. L., and E. B. Ferris; M. Berry; W. V. Whitehorn. Spontaneous pneumothorax and ascent in airplane. J.A.M.A. 127:944 (1945).

29. Ernsting, J. *In* Gillies, J. A. A Textbook of Aviation Physiology, 1st ed. New York: Pergamon Press, 1965.

30. Fischer, B. Der gutartige Spontanpneumothorax durch Ruptur von Spitzennarbenblasen, ein typisches Krankheitsbild. Mit Beitraegen zur Lehre vom Emphysem. Klin. Med. 95:1 (1922).

31. Gaensler, E. A. Parietal pleurectomy for recurrent spontaneous pneumothorax. Surg. Gynec. Obstet. 102:293 (1956).

32. Gellenthien, C. H. Altitude and artificial pneumothorax. J.A.M.A. 114:727 (1940).

33. Hamman, L. Spontaneous interstitial emphysema of lungs. Trans. Ass. Amer. Physicians 52:311 (1937).

34. Hansen, J. L. Spontaneous pneumothorax. Acta Med. Scand. 132:517 (1949).

35. Hartzell, H. C. Spontaneous hemopneumothorax: report of three cases and review of literature. Ann. Intern. Med. 17:496 (1942).

36. Hayashi, J. Ueber toedlichen Pneumothorax durch Infarkt und Emphysem. Frankfurt. Z. Path. 16:1 (1914).

37. Heath, E. M. Spontaneous pneumothorax in healthy young adults with particular reference to the etiological role of aerial ascent. Amer. J. Med. Sci. 211:138 (1946).

38. Henry, J. P. A determination of the mechanical limits to safe pressurization of the mammalian lung. Nat. Research Council, Div. Med. Sciences, Report No. 463 (1945).

39. Holter, H. V., and O. Horwitz. Spontaneous pneumothorax produced by ascent in an airplane. J.A.M.A. 127:519 (1945).

40. Hughes, F. A., N. H. Kraeft, and C. C. Lowry. Treatment of idiopathic spontaneous pneumothorax. J.A.M.A. 146:244 (1951).

41. Hyde, L. Benign spontaneous pneumothorax. Ann. Intern. Med. 56:746 (1962).

42. Jones, M. R., and H. A. Lyons. Spontaneous pneumothorax. Amer. J. Med. Sci. 277:13 (1954).

43. Joynt, G. H. C., and R. C. Laird. Treatment of spontaneous pneumothorax with kaolin. Dis. Chest 34:1 (1958).

44. Kaufman, J. L., J. M. Hanner, and I. D. Baronofsky. Spontaneous pneuomothorax. A review of experience at a large naval hospital. Western J. Surg. 66:73 (1958).

45. Kircher, L. T., and R. L. Swartzel. Spontaneous pneumothorax and its treatment. J.A.M.A. 155:24 (1954).

46. Kjaergaard, H. Spontaneous pneumothorax in the apparently healthy. Acta Med. Scand. 43:1 (1932).

47. Klassen, K. P., and C. V. Meckstroth. Treatment of spontaneous pneumothorax. Prompt expansion with controlled thoracotomy tube suction. J.A.M.A. 182:1 (1962).

48. Knoepp, L. F. The surgical considerations of bullous emphysema. Dis. Chest 40:55 (1961).

49. Knowles, J. H., R. Gorlin, and C. F. Storey. Effects of pleural talc poudrage on pulmonary function. J. Thorac. Surg. 34:250 (1957).

50. Koestler, A. G. Replication and extension of rapid decompression of chimpanzees to a near vacuum. ARL-TR-67-2, 1967; and personal communication, Mar. 1967.

51. Kolder, H. Explosive Dekompression auf Unterdruck. Die Folgen der Abnahme des Luftdruckes in kuerzester Zeit. Sitzungsberichte, Oest. Akad. Wiss., Math. Nat. Kl., pt. II, 165:357 (1956).

52. Kolder, H. Explosive Kompression. Pleotzliche Erhoehung des Luftdruckes von Unterdruck auf Normaldruck. Pflueger Arch. Ges. Physiol. 264:441 (1957).

53. Kolder, H. Explosive decompression. I. Europ. Congr. Aviat. Med., Scheveningen 1956. Aeromed. Acta 147 (special ed.) (1957).

54. Kreutzer, F. L., L. G. Brizzolara, and W. L. Rogers. Treatment of spontaneous pneumothorax by means of continuous intrapleural suction. Dis. Chest 21:663 (1952).

55. Laennec, R. T. H. De l'auscultation médiate, ou traité du diagnostic des maladies de poumons et du coeur, vol. I. Paris: J. A. Brosson and J. S. Chaudé, 1819.

56. Laurenzi, G. A., G. M. Turino, and A. P. Fishman. Bullous disease of the lung. Amer. J. Med. 32:361 (1962).

57. Leach, J. E. Pneumothorax in young adult males. Descriptive statistics in one hundred and twenty-six cases. Arch. Intern. Med. (Chicago) 76:264 (1945).

58. Lindskog, G. E., and N. A. Halasz. Spontaneous pneumothorax. A consideration of pathogenesis and management with review of seventy-two hospitalized cases. Arch. Surg. (Chicago) 75:693 (1957).

59. Lovelace, W. R., II, and H. C. Hinshaw. The hazards of aerial transportation to patients with pneumothorax. Proc. Mayo Clin. 16:40 (1941).

60. Lovelace, W. R., II, and H. C. Hinshaw. Dangers of aerial transportation to persons with pneumothorax. Roentgenographic demonstration of the effect of decreased barometric pressure (high altitude) and of increased barometric pressure. J.A.M.A. 113:1275 (1942).

61. Lovelace, W. R., II, and H. C. Hinshaw. Aerial transportation of patients – with special reference to traumatic pneumothorax diaphragmatic hernia and mediastinal emphysema. War Med. (Chicago) 2:580 (1942).

62. Lovelace, W. R., II, and J. Hargreaves. Transportation of patients by airplane. J. Aviation Med. 13:2 (1942).

63. Luft, U. C. Physiological aspects of pressure cabins and rapid decompression. *In* Handbook of respiratory physiology, ch. 8, pp. 129-142. USAF School of Aviation Medicine, 1954.

64. Lynn, R. B. Spontaneous pneumothorax. Dis. Chest 48:251 (1965).

65. Macklin, C. C. Transport of air along sheaths of pulmonary blood vessels from alveoli to mediastinum; clinical implications. Arch. Intern. Med. (Chicago) 64:913 (1939).

66. Markovits, A. S., and R. B. Phillips. Lung collapse in aviation. J.A.M.A. 164:1569 (1957).

67. Maxwell, J. The production of pleural adhesions by kaolin injection. Thorax 9:10 (1954).

68. Moser, M. Spontaneous hemopneumothorax. Treatment by early thoracocentesis. Dis. Chest 19:339 (1951).

69. Moxon, R. K. Spontaneous pneumothorax. U. S. Armed Forces Med. J. 1:1157 (1950).

70. Myers, J. A. Simple spontaneous pneumothorax. Dis. Chest 26:420 (1954).

71. Myerson, R. M. Spontaneous pneumothorax. A clinical study on one hundred consecutive cases. New Eng. J. Med. 238:461 (1948).

72. Niehaus, R. F. Simple spontaneous pneumothorax in apparently healthy individuals, 24 cases. Amer. J. Roentgen. 57:12 (1947).

73. Onesti, R., E. D'Elia, and S. Marini. Pneumotorace spontaneo idiopatico insorto in volo   Second World and Sixth European Aviation and Space Medicine Congress, Rome, 1959, vol. 2, pt. 1, pp 294-299. Rome (1961).

74. Ornstein, G. G., and L. Lercher. Spontaneous pneumothorax in apparently healthy individuals—A clinical study of fifty-eight cases with a discussion of the pathogenesis. Quart. Bull. Sea View Hosp. 7:149 (1942).

75. Parker, G. W., and R. B. Stonehill. Further considerations of the roentgenologic evaluation of flying personnel at simulated altitude. Aerospace Med. 32:501 (1961).

76. Paul, J. S., E. J. Beattie, Jr., and B. Blades. Lung function studies in poudrage treatment of recurrent pneumothorax. J. Thorac. Surg. 22:52 (1951).

77. Pease, P. P., L. G. Steuer, and A. S. Chapman. Spontaneous pneumothorax in soldiers. Bull. US Army Med. Dep. 82:102 (1944).

78. Perry, K. M. A. On spontaneous pneumothorax. Quart. J. Med. 8:1 (1939).

79. Polak, B. and H. Adams. Traumatic air embolism in submarine escape training. US Naval Med. Bull. 30:165 (1932).

80. Rahn, H., and L. E. Farhi. Gaseous environment and atelectasis. Fed. Proc. 22:1035 (1963).

81. Rottenberg, L. A., and R. Golden. Spontaneous pneumothorax: A study of 105 cases. Radiology 53:157 (1949).

82. Santos, C., and F. Tanchanco, Jr. Clinical aspects and incidence of spontaneous pneumothorax in 49,198 fluoroscopic examinations. Bull. Quezon Inst. 1:213 (1940). (Abstract) Radiology 37:118 (1941).

83. Sattler, A. Pathogenesis of so-called idiopathic spontaneous pneumothorax and related conditions. Dis. Chest. 21:315 (1952).

84. Schneider, L., and I. I. Reissman. Idiopathic spontaneous pneumothorax: History of 100 unselected cases. Radiology 44:485 (1945).

85. Spontaneous pneumothorax. Brit. Med. J. 1:1262 (1965).

86. Stein, G. H., E. B. McConkie, and A. J. Kuehn. Spontaneous pneumothorax. War Med. (Chicago) 4:324 (1943).

87. Stonehill, R. B., J. E. Reed, and G. W. Parker. Roentgenologic evaluation of flying personnel at simulated altitude by use of portable equipment. Aerospace Med. 30:751 (1959).

88. Tillisch, J. H., and E. T. Carter. Clinical problems in aviation medicine. Case report No. 3. Aerospace Med. 32:1068 (1961).

89. Thomas, P. A. Spontaneous pneumothorax—Modern concepts in etiology and treatment of an important syndrome in military practice. Milit. Med. 124:116 (1959).

90. United States Air Force, Office of the Surgeon General, 1st to 11th Annual Report of the USAF Medical Service, 1952 to 1964.

91. United States Air Force, Medical Service. Physiological Technician's Training Manual, AFM 160-5, 6-6 (1965).

92. Van Allen, C. M., G. E. Lindskog, and H. S. Richter. Gaseous interchange between adjacent lung lobules. Yale J. Biol. Med. 2:297 (1930).

93. Watkins, E., Jr., and A. C. Hering. Indications for surgical intervention in "spontaneous" pneumothorax. Surg. Clin. N. Amer. 41:709 (1961).

94. Wood, E. A., A. C. Nolan, D. E. Donald, and L. Cronin. Influence of acceleration on pulmonary physiology. Fed. Proc. 22:1024 (1963).

95. Wood, E. H. Some effects of gravitational and inertial forces on the cardiopulmonary system. Aerospace Med. 38:225 (1967).

96. York, E. Post-flight discomfort in aviators: Aero-atelectasis. Aerospace Med. 38:192 (1967).

97. Zarriello, J. J., and J. J. Acker. Spontaneous pneumothorax in flight: A case report and a brief review of the literature. Aerospace Med. 30:418 (1959).